Excerpt from AANA learning module

Hitt Medical Writing

By Shannon Aymes, MD

Acute renal failure is defined as an acute decrease in glomerular filtration rate, resulting in increased creatinine, blood urea nitrogen, and alterations in electrolyte balance. Patients with a history of hypertension, chronic kidney disease, diabetes, over 55 years of age, liver disease, and hypovolemia are at greatest risk for developing postoperative acute renal failure. The risk is further heightened as the patient is exposed to contrast agents, antibiotics, and nonsteroidal anti-inflammatory medications.

The differential diagnosis of acute renal failure can be divided into three categories: prerenal failure, intrinsic renal failure, and postrenal failure.

Prerenal failure is a result of decrease blood flow to the kidneys. Intrinsic renal failure results from direct damage to the kidney at the level of the glomeruli, tubules, vasculature, or interstitial tissue. Finally, post-renal failure is a result of mechanical obstruction, commonly from prostate enlargement, nephrolithiasis, and malignancy.

Administration of anesthesia can directly and indirectly affect renal function. The circulatory system may be impacted, resulting in decreased perfusion or increased vascular resistance. Tachycardia may be a result of anesthesia on the cardiovascular system. Similarly, a decrease in venous return is associated with spinal and epidural anesthesia. Despite autoregulatory mechanisms within the kidney, anesthesia may lower renal blood flow by 30 to 40 percent. Anesthetic agents can also be directly nephrotoxic through mechanisms related to fluoride ion toxicity. Fluoride interferes with the sodium and chloride transport channel by causing vasodilation and inhibiting antidiuretic hormone. ADH inhibition will lead to decreased reabsorption of water. Hypernatremia, polyuria, hyperosmolality, and rising BUN and creatinine are associated with fluoride ion toxicity.

Many of the medications commonly used during surgical procedures are partially excreted by the kidneys. A detailed understanding of their mechanisms of action and interactions with renal impairment is of vital importance. Both propofol and etomidate have been shown to have heightened pharmacological effects in the setting of renal failure. This is thought to be associated with hypoalbuminemia and lower protein binding. Benzodiazepines, although metabolized by the liver, are excreted by the kidneys. Thus, diazepam and midazolam may accumulate in renal failure and in hypoalbuminemia. In the setting of renal disease, patients exposed to barbiturates may show a lower threshold to the medication during the induction process. Accumulation of metabolites of both ketamine and the opioids, morphine and meperidine, is associated with renal insufficiency.